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Authors' response

From CHARLES POOLE,¹ ULRIKE PETERS,² DORA IL'YASOVA³ and LENORE ARAB⁴

Dr Tokudome and colleagues¹ ask for more than any systematic review of the currently available epidemiological literature on tea and cardiovascular disease can deliver. If a sufficient number of investigators were to publish trend estimates for specific cardiovascular disease outcomes in relation to intake of specific compounds or classes of compounds in tea and other beverages and foods, those estimates would be able to be systematically reviewed. Until then, it is a hypothesis in search of a literature.

Very few populations contain sufficient numbers of people drinking ≥ 10 cups of tea per day to permit effects of intakes that high to be estimated with anything but the grossest imprecision. We evaluated trend estimates at an increment of three cups per day^{2,3} because that was the largest increment that fit well within the observed ranges of actual intakes in the published study populations.

Regarding potential interventions that might conceivably result someday from research on this topic, an increase of three cups per day in average tea intake would be a wildly unrealistic goal to set at the population level. Getting a population to increase its average tea intake by 10 cups per day would be pure fantasy. There is always the possibility that one or more preventive compounds might be identified and used for nutritional supplementation or dietary fortification, but the realization of such a possibility is a very long way off at best.

As a more general matter, Dr Tokudome and colleagues suggest that statistical significance constitutes proof. It does not.

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A hypothesis on the sexual behaviour of men who are destined to develop prostate cancer

From WILLIAM H JAMES

There is general agreement that prostatic cancer (PC) has genetic and environmental determinants. However, the putative susceptibility genes have so far largely evaded identification¹ and the environmental factors are also proving difficult to unravel. But for many years, attention has focussed on the possibility of

endocrine (particularly androgenic) determinants of one sort or another. The difficulty is that though it is accepted that androgens stimulate PC *in vitro* and *in vivo*, direct data on circulating concentrations of steroids are equivocal.^{2–4} However, men who are destined to develop the cancer reportedly have a statistically significant excess of sons,⁵ and there are good grounds for supposing that that indicates high androgen levels at the time of conception.^{6,7} Frequently these conceptions

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